

Risk Analysis for Terrestrial Wildlife

Potential risk to terrestrial wildlife (i.e., birds and mammals) resulting from exposure to substances of concern (SOCs) associated with Onondaga Lake will be evaluated using a food web modeling approach. This assessment will begin from the results of the screening risk assessment and progress to a site-specific baseline ecological risk assessment (BERA) by replacing the inherently conservative screening assumptions with site-specific data and observations. This memorandum is intended to partially fulfill the requirements of Steps 3 through 6 of the ecological risk assessment process as outlined by the U.S. Environmental Protection Agency (EPA) (U.S. EPA 1997) and provide a discussion basis for the remaining scientific/management decision points (SMDP).

The SMDP associated with the screening-level risk assessment will involve the selection of the contaminants of concern (COCs) and receptors that will be considered in the BERA, and will be based on the findings of the screening assessment. This subset of SOCs and specific exposure pathways will be subjected to a BERA as detailed in Steps 3 through 7 of U.S. EPA (1997). Steps 5 and 6 deal almost exclusively with issues related to practical site sampling and, therefore, do not directly involve the

development of the models necessary to characterize the risk to terrestrial receptors. Specific sections of the ecological risk assessment guidance (U.S. EPA 1997) that will be addressed are discussed below.

Baseline Risk Assessment Problem Formulation

Step 3 in the development of the BERA is problem formulation. This is similar to the preliminary problem formulation process carried out in Step 1, but will be performed in much greater detail. The problem formulation in Step 3 establishes the goals, breadth, and focus of the BERA. It also establishes the assessment endpoints, and the specific ecological values to be protected (U.S. EPA 1997).

The baseline problem formulation will begin from a refinement of the preliminary COCs (Section 3.2 of EPA's guidance [U.S. EPA 1997]). This will be determined predominantly from the results of the screening ecological risk assessment (Step 2) and negotiations between the New York State Department of Environmental Conservation (NYSDEC) and Honeywell.

Contaminant Transport and Fate, Ecosystems Potentially at Risk, and Complete Exposure Pathways

After the list of SOC's and receptor exposure pathways has been finalized, the potential exposure pathways will be re-examined and refined. This will involve more specific considerations of the SOC's transport and fate, both natural and anthropogenic. The principal locations of accumulation will be identified as well as any secondary media of potential exposure. Points of potential bioaccumulation will also be identified along with information pertaining to bioavailability. Information on SOC transport and fate will be determined from the analysis of onsite data as well as from appropriate literature sources. Currently, the primary exposure routes for SOC exposure are soil/sediment, surface water, and prey items.

Concurrent with the analysis of potential transport and fate issues, the specific ecosystems potentially at risk will also be identified. Based on past direction from NYSDEC (NYSDEC 1999), the risk to terrestrial receptors will be determined for the following habitat types associated with Onondaga Lake:

- Onondaga Lake proper consisting of a lacustrine environment containing both pelagic and littoral habitat available to receptors native to upstate New York
- Littoral wetlands surrounding Onondaga Lake and designated SYW-6, SYW-10, SYW-12 and SYW-19
- Uplands associated with the dredge spoils located south of SYW-6.

The exposure pathways that will be considered in the BERA will involve the primary pathways by which SOC's present in the sediments, water column, and biota of Onondaga Lake may contact the ecological receptors that are at greatest risk. In general, the exposure pathways considered for assessment will be similar in structure to those evaluated in the screening risk assessment, that is, potential SOC exposure through the ingestion of prey, drinking water, and soil or sediment.

Selection of Assessment Endpoints

Ecological risk assessment involves the identification of potential impacts to numerous species that are likely to be exposed at differing degrees, or are likely to respond differently to SOC's present in a given location. It is not practical or necessary to directly evaluate risk to all of the individual components of the exposed ecosystems. Assessment endpoints are intended to focus the risk assessment on those specific components most likely to be at risk. Therefore, the assessment will examine specific receptors considered to be most at risk by virtue of their exposure to the highest concentrations of specific SOC's. For the assessment of terrestrial receptors, the primary assessment endpoint is proposed to be the sustainability of indigenous wildlife populations.

A second management endpoint that will be examined is the 95 percent upper confidence limit (UCL) on the mean of the hazard quotient calculated for each of the species assessed. This is being performed at the request of NYSDEC (NYSDEC 2000).

Conceptual Model and Risk Questions

The conceptual site model establishes the complete exposure pathways that will be evaluated in the ecological risk assessment. From these pathways, risk questions will be developed to address measurement endpoints. The habitat-specific conceptual models and associated risk questions will be as follows:

Onondaga Lake (pelagic)—The receptors at greatest risk in this habitat are those that forage within the water column of the open lake. There are no mammalian species indigenous to this region that utilize this habitat. However, both the osprey and the double-crested cormorant do hunt in the pelagic zone, and therefore may be exposed to SOC_s in this region of Onondaga Lake. Therefore, the measurement endpoints for the terrestrial ecological risk assessment for the pelagic zone will be the proportion of the exposed subpopulations of osprey and double-crested cormorant whose exposure exceeds that considered to be an acceptable threshold, and the resulting impact on the sustainability of the subpopulations. The risk questions associated with these endpoints therefore will be: 1) what proportion of the subpopulation receives an exposure greater than the

toxicity reference value (TRV), and 2) is that proportion sufficient to adversely affect population sustainability?

Onondaga Lake (littoral/wetland)—The receptors at greatest risk in this habitat are those that forage on or near the shore of the lake and are dependent upon indigenous aquatic organisms as their primary food source. The terrestrial receptors considered most likely to be at risk are mink, river otter, belted kingfisher, mallard, little brown bat, tree swallow, and great blue heron. Therefore, the measurement endpoints for the terrestrial ecological risk assessment for the littoral and wetland zones will be the proportion of the exposed subpopulations of the above receptors whose exposure exceeds an acceptable threshold, and the resulting impact on the sustainability of the subpopulations. Therefore, the risk questions associated with these endpoints will be: 1) what proportion of the subpopulation receives exposure greater than the TRV, and 2) is that proportion sufficient to adversely affect the sustainability of the subpopulation?

Uplands Dredge Spoils—The receptors at greatest risk in this habitat are those that forage on insects and small mammals indigenous to the region. Those at greatest risk through this pathway are the short-tailed shrew (native

insectivore) and the red-tailed hawk (native top carnivore). Therefore, the measurement endpoints for the terrestrial ecological risk assessment for the uplands dredge spoils will be the proportion of the exposed subpopulations of the above receptors whose exposure exceeds an acceptable threshold, and the resulting impact on the sustainability of the subpopulations. The risk questions associated with these endpoints therefore will be: 1) what proportion of the subpopulation receives an exposure greater than the TRV, and 2) is that proportion sufficient to adversely impact the sustainability of the subpopulation?

Characterizing Exposures

Exposure will be determined by modeling the co-occurrence of the SOC_s with the physical presence of the receptors in both space and time. In order to do this, both the stressor and ecosystem will be characterized on similar temporal and spatial scales. Stressor characterization that describes the distribution patterns of the retained SOC_s within the potentially affected habitats will be based on available site-specific data. SOC concentrations in potential media of exposure will be determined from site-specific data. Receptor-specific interactions will be predicted based on the best accounts of the receptor's life history in available literature.

Because of the complexity and potential uncertainty associated with these estimates, the exposure assessment will use a tiered methodology that will look at various exposure scenarios based on the receptors' behaviors. This will begin with the conservative exposure estimates derived in the screening risk assessment and will progressively examine the effects of life cycle and behavior on potential exposures and impacts. The specific scenarios that will be tested will be as follows:

1. Exposure of the specific receptors to all retained SOC's assuming that, while in the general vicinity of Onondaga Lake, their entire dietary, drinking water, and incidental sediment ingestion is derived from the assessment area. The temporal analysis will be performed in accordance with the method outlined below as per direction from NYSDEC (NYSDEC 2000). This tier is intended to demonstrate the worst-case exposure and although an unrealistic scenario for many of the receptors under consideration, this level of analysis will provide a basis for later uncertainty analysis.

2. Exposure of specific receptors to all retained SOC's assuming that, while in the general vicinity of Onondaga Lake, the receptors home range will be centered on the western shore of the lake. This scenario is tantamount to assuming that the receptors will be nesting within the assessment area. Because none of the habitats under consideration is physically adequate to support double-crested cormorant nesting (i.e., no isolated islands), that receptor will be excluded from this level of analysis. All other receptors and habitats will be evaluated.
3. Exposure of specific receptors to all retained SOC's using site-specific data on known nesting and foraging locations. This level of analysis is intended to reflect the potential risks within the situation that actually existed in 1992. For receptors with no available data on home range locations, the assumption of closest home range location will be conservatively assumed to be the same as that used in Scenario 2.

Representative ingestion rates will be determined from available literature sources. Rates of SOC exposure will then be estimated based on the concentration of SOCs in the media and the rate of media intake by the receptor. The model that will be used to estimate the rate of SOC exposure will be structured to predict the site-specific daily doses to the receptors. This will allow for a direct comparison of exposure to toxicity in the risk characterization. The general structure of the model used to estimate the exposure rate for a given chemical by a wildlife receptor will be as follows:

where:

EER_A = estimated exposure rate at a given location A (mg/kg body weight-day)

n = individual receptor of the exposed subpopulation N

IR_p = receptor-specific prey intake rate (kg dry weight/kg body weight)

IR_w = receptor-specific water intake rate (L/kg body weight)

IR_s = receptor-specific incidental sediment intake rate (kg dry weight/kg body weight)

$[COC]_p$ = representative COC concentration in the receptor's prey (mg/kg dry weight)

$[COC]_w$ = representative COC concentration in the receptor's drinking water (mg/L)

$[COC]_s$ = representative COC concentration in the sediments incidentally ingested (mg/kg dry weight)

Exposure concentrations will be determined from a synthesis of site-specific data and the life cycle behavior of the receptors. Specific estimates of SOC concentrations in consumed media will be determined based on estimates of the likelihood that a receptor would consume a given concentration. This will be determined as follows:

where:

x = potential medium of exposure (prey, drinking water, soil/sediment)

$[SOC]_x$ = estimated receptor-specific concentration of SOC in
medium x (mg/kg dry weight)

a = specific measured sample from medium x

$[SOC]_a$ = observed SOC concentration in sample a (mg/kg dry
weight)

$L(EXP)$ = likelihood of receptor exposure to sample a (unitless)

For piscivores, the likelihood of their exposure at a given concentration of an SOC will be based on the proportion of the diet composed of specific sizes of fish. The specific behavioral assumptions that will be used to represent these likelihoods are listed in Revised Table 1. SOC exposure through incidental ingestion of sediment or soil will be modeled based on the range of concentrations measured within the assessment units and directly correlated with the distribution of the SOC within the prey. This is a conservative approach since it automatically assumes that exposure to high concentrations of SOC in the prey will be associated with exposure to high SOC concentrations in the soil/sediment. No selection assumption will be applied for drinking water within an assessment unit. Such exposure to any particular measured concentration of an SOC will be considered uniform and random. Total exposure concentrations will be determined by summing the contributions from all sources from site-specific data. The

exposure rates will be expressed as a probability density function. The 95 percent UCL for the mean will be determined, as requested by NYSDEC (NYSDEC 2000).

The distribution of exposures for the receptor subpopulations will be determined through the summation of exposure across all possible locations, and weighted based on the probability that a proportion of the exposed subpopulations will use a given location as foraging habitat. To accomplish this, the following algorithms will be applied:

where:

EER_{Total} = total estimated exposure rate (mg/kg body weight-day)

EEC_{Bkgnd} = estimated exposure rate based on background estimates of
COC concentrations (mg/kg body weight-day)

$EEC_{Site A}$ = estimated exposure rate based on the estimates of COC
concentrations within the assessment units (mg/kg
body weight-day)

P_{not-NY} = proportion of time spent outside the region of Onondaga
Lake (unitless)

P_{NY} = proportion of time spent in the region of Onondaga Lake
(unitless)

$P_{Site A}$ = proportion of time spent at any given assessment unit A
(unitless)

$P_{Offsite}$ = proportion of time spent in the region of Onondaga Lake,
but not associated with any assessment unit (unitless)

Life history parameters will be determined from the most appropriate literature resources and provided in detail within the BERA. Proposed values and sources are listed in Revised Table 2. Specifics for their derivation and use in the risk models are as follows:

Body Weights—Body weight estimates were determined from literature reports. Median values representative of populations indigenous to the northeastern United States were preferred.

Media Intake Values—Intake rates for food and water were taken from reported literature values. When not available, allometric scaling was used based on the reported body weights. For food intake, the scaling relation of Nagy (1987) was applied to receptors with the exception of the great blue heron. For this species, a wading bird-specific scaling relation developed by Kushlan (1978) was applied. The intake rate for the tree swallow is currently under review. For estimates of drinking water intake, the allometric relation of Calder and Braun (1983) was applied to all receptors. Information for incidental soil or sediment ingestion is only available for the mallard (U.S. EPA 1993). For all other receptors exposed to soil or sediment through ingestion, the ingestion rates will be determined from the limited available published data, using best professional judgment to match reported species with the receptors. An uncertainty factor of less than or equal to 2 (not to exceed a total sediment ingestion rate of 30 percent of total dietary intake) was applied to derive the uniform distribution.

Migration Cycles—Because of concerns expressed by NYSDEC (NYSDEC 1999, 2000) with regard to the timescale associated with this level of behavior, proportion of time spent onsite will be determined within the context of the exposure period from which the TRV was derived. This temporal adjustment will be applied only to the migratory species. For these receptors, the minimum duration of exposure will be defined as that period observed in the toxicological study between the commencement of treatment and the first reported observation of an adverse effect. For the receptors associated with Onondaga Lake, this treatment period will be conservatively assumed to commence with the receptors arrival at Onondaga Lake. If the study provides a period greater than or equal to 1 year, then the exposure is assumed to occur within 365 days. The location of the receptors within the migratory cycle will be determined from literature reports using the closest available location to upstate New York. The proportion of time spent in the vicinity of Onondaga Lake (P_{NY}) versus elsewhere on their migratory cycles will be determined as follows:

where:

P_{NY} = proportion of time spent in the region of Onondaga Lake
(unitless)

$P_{\text{not-}NY}$ = proportion of time spent outside the region of Onondaga
lake (unitless)

T_{depart} = estimated time when receptor departs the Onondaga Lake
region (annual days)

T_{arrive} = estimated time when receptor arrives in the Onondaga
Lake region (annual days)

t_{study} = duration between start of treatment and manifestation of
adverse effects in the study used to derive the TRV
(days)

Reported estimates or life cycle scheduling will be given a central date bounded by 15-days of uniform uncertainty (for example, mid-March will be evaluated as March 1st through March 30th). Ranges of dates will be used as reported, assuming uniform uncertainty. For nonmigratory birds

and mammals, no temporal consideration will be included in the exposure models.

Foraging Area—The implications of foraging area will be considered only for those receptors whose life histories indicate that they forage over a typical range greater than the total habitat represented by Onondaga Lake. This level of analysis will only be applied in the Tier 2 and Tier 3 analyses. For Tier 2, the foraging area will be centered on Onondaga Lake. For Tier 3, the foraging area will be centered on the closest positively identified nesting, roosting or burrowing site. Two hypothetical circles representing the mean and maximum foraging radius will describe the foraging area. The radii of these circles will be determined from the scientific literature and will represent the best available estimate of the mean and maximum foraging ranges, based on the type of habitat and regional location represented by Onondaga Lake. Because of the energetic requirements involved in foraging at increasing distances from the origin, a probability of habitat use will be assigned such that the mean radii will represent a cumulative probability of use of 0.5, and the maximum radii a cumulative probability of use of greater than 0.99. The interstitial probability will be assigned based on the following exponential decay function and determined by the

regression of the cumulative distribution of probabilities using the assigned radii and a value of 1.0 for the origin (i.e., assumed nesting location).

where:

P_r = probability based on distance from the origin (unitless)

a = dependent asymptote (unitless)

b = decay slope (km^{-1})

r = radial distance from the origin (km)

In order to account for variations in habitat availability, the density of usable foraging, as determined by a geographic information system analysis, was overlaid on the radial probability. This analysis weights the receptor distribution to select high-density habitat (such as the lake itself) in preference to isolated streams and rivers. Habitat density was assumed to be independent from the origin and therefore was modeled as follows:

where:

$P_{(x,y)}$ = probability of foraging use (unitless)

$H_{(x,y)}$ = habitat density at location x,y (m^2 or m)

H_{total} = total habitat density at all locations within the foraging
area (m^2 or m)

P_r = probability based on distance from the origin (unitless)

Probability of exposure to any of the assessment sites (Onondaga Lake, Willis Avenue Site, Semet Residue Ponds, LCP Bridge Street Site, Geddes Brook and Ninemile Creek, Waste Beds 1–15, East Flume, etc.) will be determined based on the proportion of the foraging range they constitute, and weighted based on the probability of use by the receptor population. This will constitute a probability density that will be substituted into the risk model as $P_{Site\ a}$. Probability of exposure to background concentrations offsite will be defined as the remainder probability ($1 - P_{Site\ a}$).

Background Concentrations—Background concentrations of SOC_s are intended to simulate potential receptor exposures when they are not associated with a site for which SOC concentrations have been measured. Estimations of background concentrations will be based on available scientific literature. Preference will be given to EPA and NYSDEC surveys of areas specifically

not identified as being contaminated. If such reports are not available, then appropriate values will be determined from the scientific literature.

Characterizing Ecological Effects

The exposure-response analysis describes the relationship between the magnitude, frequency, and duration of exposure to an SOC, and the magnitude of the expected response. For terrestrial receptors exposed to SOCs from Onondaga Lake, there are no data pertaining to any observed adverse impact resulting from onsite exposures to SOCs. Therefore, as permitted in the ecological risk assessment guidance for Superfund (U.S. EPA 1997), a threshold for adverse effects will be developed for the BERA using available literature and site-specific information in place of conservative assumptions applied in the screening assessment.

The response of individual members of the exposed population to SOC exposure will be determined by comparing rates of exposure to TRVs. These TRVs will be derived from a survey of available ecotoxicological literature. Evaluation of applicability will be based on the comparability of the reference study to the situations present on Onondaga Lake. The values selected will be those from studies that best mirror the specific type

and condition of the receptor, and describe the most sensitive toxicological endpoint that would be expected to directly impact the measurement endpoints described for the BERA.

Uncertainty associated with the response estimates will be addressed in the food web models. This will be accomplished by assigning various levels of uncertainty with regard to extrapolations involving interspecies variations, subchronic to chronic uncertainty and lowest-observed-adverse-effect level to no-observed-adverse-effect level extrapolations. Guidance for the derivation of these levels of uncertainty, in order of priority, will be as follows:

- 1. NYSDEC (1997) Ambient Water Quality for Protection of Wildlife*
- 2. Great Lakes Water Quality Initiative Criteria Documents for the Protection of Wildlife (U.S. EPA 1995)*
- 3. Performing Ecological Risk Assessments (Calabrese and Baldwin 1993)*

Proposed TRVs and levels of uncertainty are provided in Revised Table 3.

Risk Estimation

Risk estimations will be expressed in two fashions. First, an estimate of hazard quotients will be reported for the 95 percent UCL for the mean exposure rate of the exposed receptor subpopulations. The second will be the estimate of the proportion of the exposed subpopulation that would be expected to receive an exposure of the SOC at a rate greater than that purported to result in an adverse impact. This will be defined as that portion of the population whose projected exposure exceeds the TRV. This impact estimate will then form the basis for projecting potential population stabilities in the final component of this analysis.

The estimate of the 95 percent UCL for the mean will be reported as requested by NYSDEC (NYSDEC 2000). This estimate will be based on the pooled standard error for all parameter inputs in the above-described models, and will be reported as variance to the estimate of hazard quotient for all three tiers of analyses. Estimates of the hazard quotients for the mean exposure to background SOC concentrations for the same receptor populations will also be provided.

The risk to terrestrial wildlife will also be expressed proportionally, as the percentage of the exposed subpopulation whose SOC exposure exceeds the TRV. This estimate will be determined based on the probable level of exposure as determined by the food web model described above. Estimates will be reported for all three tiers of analysis along with the risk estimates associated with background exposures. Comparisons between risk due to exposure to background concentrations and risk associated with the presence and use of Onondaga Lake as well as the other assessment units will be reported as the difference between the results of the food web model and the risk associated with background exposures. Potential impacts due to site-related SOC's will be used to evaluate the influence of SOC exposure on population sustainability.

Estimates of impacts on population sustainability due to SOC's will be determined by comparing the proportion of the exposed subpopulations at risk (i.e., predicted to experience exposures greater than the TRV) to the reproductive capacity for the specific receptor. The estimation of the reproductive capacity for the receptor subpopulations will be determined using cohort modeling. This approach balances rates of reproduction against rates of natural mortality within the context of the species' life

cycle. The population dynamics will be modeled using the following algorithms:

For species that mature in 1 year

For species that mature in 2 years

where:

P_t = population of the reproductive cohort at time t

F_{1t} = population of yearling cohort at time t

F_{2t} = population of second year cohort at time t

- r = reproductive rate for the reproductive cohort
- m_a = mortality rate for the reproductive cohort
- m_{F1} = mortality rate for the yearling cohort
- m_{F2} = mortality rate for the second year cohort
- E_t = emigration of reproductive individuals in excess of carrying capacity at time t

If the rate of reproduction is equal to or greater than the rate of mortality, then the subpopulation can be assumed to be stable, since it possesses the potential to maintain the population level of the reproductive cohort at the carrying capacity. However, if the rate of mortality exceeds the rate of reproduction, then the exposed subpopulation will become reliant on immigration from non-exposed subpopulations in order to maintain the population level of the reproductive cohort. In this situation, the exposed subpopulation can be said to be unstable. This is a conservative approach since density-dependent variations in survivorship are not included.

The results of the cohort model will be synthesized with the results of the risk analysis by assuming that the proportion of the population whose exposure exceeded the TRV will either be subtracted from the reproductive rates of the population or added to the mortality rate, depending on the

type of adverse effect associated with the TRV. Because this requires the determination of the proportional impact relative to the TRV, the conservative assumptions of complete adverse response will be used. For example, if the TRV is based on an observed reduction in reproduction, then all individuals whose exposure exceeded the TRV will be assumed to suffer complete reproductive failure. For non-reproductive endpoints, exceedance of the TRV will be assumed to result in the individual's mortality. Therefore, the evaluation of the input parameters specific to Onondaga Lake will be as follows:

For reproduction:

For non-reproduction:

where:

r_{OL} = reproductive rate for subpopulations exposed to Onondaga Lake

r_{bkgnd} = natural rate of reproduction for the receptor species

r_{COC} = reproductive impact as the result of exposure to a specific
SOC

m_{OL} = mortality rate for subpopulations exposed to Onondaga
Lake

m_{bkgnd} = natural rate of mortality for the receptor species (cohort
specific)

m_{COC} = mortality impact as the result of exposure to a specific SOC

If this results in a probability greater than 0 that the overall mortality may exceed the rate of reproduction for the exposed subpopulation, then this subpopulation will be judged to have been rendered unstable as a result of the presence of an SOC in Onondaga Lake. Otherwise, it will be concluded that the presence of the SOC would not affect the sustainability of the exposed subpopulation.

Uncertainty Analysis

The uncertainty analysis will consist of two sections. The first will be a quantitative analysis of the uncertainties associated with the parameters used in the food web models. This will be expressed as percent attribution to the variance. The quantitative analysis will be limited to those parameters for which the degrees of uncertainty have been identified and will include such inputs as onsite SOC concentrations as well as TRV and temporal use probabilities for which levels of uncertainty have been deliberately introduced. The parameters most responsible for unaccounted variance will be identified and discussed in context with the models' predictions.

The second section of the uncertainty analysis will involve a qualitative discussion of uncertainties that cannot be quantified in the food web model.

It will also include a discussion on the overall uncertainty associated with the predictive nature of the risk analysis.

References

Alexander, G.R. 1977. Food of vertebrate predators in trout waters in north central lower Michigan. Michigan Academ. 10:181-195.

Aulerich, R.J., and R.K. Ringer. 1977. Current status of PCB toxicity to mink, and effect on their reproduction. *Arch. Environ. Contam. Toxicol.* 6:279-292.

Bedard et al. (1995), as cited by Hatch and Weseloh 1999

Beyer, W.N., E.E. Connor, and S. Gerould. 1994. Estimates of soil ingestion by wildlife. *J. Wildl. Manage.* 58(2):375-382.

Brooks, R.P., and W.J. Davis. 1987. Habitat selection by breeding belted kingfishers (*Ceryle alcyon*). *Am. Midl. Nat.* 117(1):63-70.

Brown, L., and D. Amadon. 1968. Eagles, hawks, and falcons of the world. Volume 1. McGraw-Hill, New York, NY.

Calder, III, W.A., and E.J. Braun. 1983. Scaling of osmotic regulation in mammals and birds. *Am. J. Physiol.* 244:R601-R606.

Campo, J.J., B.C. Thompson, J.C. Barron, et al. 1993. Diet of Double-Crested Cormorants Wintering in Texas. *J. Field Ornithol.* 64(2):135-144

Charbonneau, S.M., I.C. Munro, E.A. Nera, F.A.J. Armstrong, R.F. Willes, F. Bryce, R.F. Nelson, and A. Svenson. 1976. Chronic toxicity of methylmercury in the adult cat. Interim report. *Toxicology* 5:337-349

Craighead, J.J., and F.C. Craighead. 1956. Hawks, owls, and wildlife. The Stackpole Co., Harrisburg, PA, and Wildl. Manage. Inst., Washington, DC.

Dowd, E.M., and L.D. Flake. 1985. Foraging habitats and movements of nesting great blue herons in a prairie river ecosystem, South Dakota. *J. Field Ornithol.* 56(4):379-387.

Eagle, T.C., and J.S. Whitman. 1987. Mink. pp. 615-624. In: *Wild Furbearer Management and Conservation in North America*. M. Novak, J.A. Baker, M.E. Obbard, and B. Malloch (eds). Ministry of Natural Resources, Ontario.

Fitzhugh, O.G. 1948. Use of DDT insecticides on food products. *Ind. Eng. Chem.* 40:704-705.

Fleming, W.J., M.A. Ross McLane, and E. Cromartie. 1982. Endrin decreases screech owl productivity. *J. Wildl. Manage.* 46:462-468.

Good, E., and G.W. Ware. 1969. Effects of insecticides on reproduction in the laboratory mouse: IV. Endrin and dieldrin. *Toxicol. Appl. Pharmacol.* 14:201-203.

Hartman, F.A. 1961. Locomotor mechanisms in birds. *Smithsonian Misc. Coll.* 143.

Haseltine, S.D., L. Sileo, D.J. Hoffman, and B.D. Mulhern. Unpublished. Toxicological benchmarks for wildlife: 1996 Revision. Risk Assessment Program, Health Sciences Research Division, Oak Ridge, TN. (not seen, as cited in Sample et al. 1996)

Hatch, J.J. and D.V. Weseloh. 1999. Double-crested cormorant (*Phalacrocorax auritus*). In: *The Birds of North America*. No. 441. A. Poole and F. Gill (eds). The Birds of North America, Inc., Philadelphia, PA.

Heath, R.G., J.W. Spann, and J.F. Kreitzer. 1969. Marked DDE impairment of mallard reproduction in controlled studies. *Nature* 224:47-48.

Heinz, G.H. 1979. Methylmercury: reproductive and behavioral effects on three generations of mallard ducks. *J. Wildl. Manage.* 43(2):394-401.

Kushlan, J.A. 1978. Feeding ecology of wading birds. pp. 249-296. In: *Wading Birds*. A. Sprunt, J. Ogden, and S. Winckler (eds). National Audubon Society, New York, NY.

Lamb, IV, J.C., R.E. Chapin, J. Teague, A.D. Lawton, and J.R. Reel. 1987. Reproductive effects of four phthalic acid esters in the mouse. *Toxicol. Appl. Pharmacol.* 88:255-269.

Mackenzie, R.D., R.U. Byerrum, C.F. Decker, C.A. Hoppert, and R.F. Langham. 1958. Chronic toxicity studies. II: hexavalent and trivalent chromium administered in drinking water to rats. *Am. Med. Assoc. Arch. Ind. Health* 18:232-234.

McCarty, J.P. 1995. Effects of short-term changes in environmental conditions on the foraging ecology and reproductive success of tree swallows, *Tachycineta bicolor*. Thesis. Cornell University.

McLane, M.A., and D.L. Hughes. 1980. Reproductive success of screech owls fed Aroclor® 1248. *Arch. Environ. Contam. Toxicol.* 9:661-665.

Melquist, W.E., and A.E. Dronkert. 1987. River otter. pp. 627-641. In: *Wild Furbearer Management and Conservation in North America*. M. Novak, J.A. Baker, M.E. Obbard, and B. Malloch (eds). Ministry of Natural Resources, Ontario.

Mitchell, J.L. 1961. Mink movements and populations on a Montana river. *J. Wildl. Manage.* 25:48-54.

Murray, F.J., F.A. Smith, K.D. Nitschke, C.G. Humiston, R.J. Kociba, and B.A. 1979. Three-generation reproduction study of rats given 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in the diet. *Toxicol. Appl. Pharmacol.* 50:241-251.

Nagy, K.A. 1987. Field metabolic rate and food requirement scaling in mammals and birds. *Ecol. Monogr.* 57(2):111-128.

Nelson, A.L., and A.C. Martin. 1953. Gamebird weights. *J. Wildl. Manage.* 17(1):36-42.

Nosek, A., S.R. Craven, J.R. Sullivan, S.S. Hurley, and R.E. Peterson. 1992. Toxicity and reproductive effects of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in ring-necked pheasants. *J. Toxicol. Environ. Health* 35:187-198.

NYSDEC. 1997. New York State wildlife fact sheet: Ambient water quality value for protection of wildlife. Draft. New York State Department of Environmental Conservation, Albany, NY.

NYSDEC. 1999. Letter from T. Larson, Project Manager, to A.J. Labuz, AlliedSignal Inc, Solvay, NY, regarding Onondaga Lake RI/FS draft baseline ecological risk assessment report. New York State Department of Environmental Conservation, Albany, NY.

NYSDEC. 2000. Letter from T.J. Larson, Project Manager, to A.J. Labuz, Honeywell, Syracuse, NY, dated November 17, 2000, regarding Geddes Brook/Ninemile Creek RI/FS draft

BERA report. New York State Department of Environmental Conservation, Division of Environmental Remediation, Albany, NY.

Peakall, D.B. 1974. Effects of di-N-butylphthalate and di-2-ethylhexylphthalate on the eggs of ring doves. *Bull. Environ. Contam. Toxicol.* 17:213-218.

Poole. 1984. (not seen, as cited in U.S. EPA 1993)

Preston, C.R. and R.D. Beane. 1993. Red-tailed hawk (*Buteo jamaicensis*).

In: *The Birds of North America*, No. 52. A. Poole and F. Gill (eds). The Academy of Natural Sciences, Philadelphia, PA, and The American Ornithologists' Union, Washington, DC.

Schlesinger, W.H., and G.L. Potter. 1974. Lead, copper, and cadmium Concentrations in small mammals in the Hubbard Brook Experimental Forest. *OIKOS* 25:148-152.

TAMS and Menzie-Cura. 1999. Baseline ecological risk assessment, Hudson River PCBs reassessment RI/FS. Phase 2 Report, Review copy. Books 1-3. TAMS Consultants, Inc. and Menzie-Cura and Associates, Inc.

U.S. EPA. 1993. Wildlife exposure factors handbook. Volume I.

EPA-600-R-93-187. U.S. Environmental Protection Agency, Washington, DC.

U.S. EPA. 1995. Great Lakes water quality initiative criteria documents for the protection of wildlife. EPA-820-B-95-008. U.S. Environmental Protection Agency, Office of Water, Washington, DC.

U.S. EPA. 1997. Ecological risk assessment guidance for Superfund: Process for designing and conducting ecological risk assessments. Interim Final. U.S. Environmental Protection Agency, Environmental Response Team, Edison, NJ.

Van Daele, L.J., and H.A. Van Daele. 1982. Factors affecting the productivity of ospreys nesting in west-central Idaho. *Condor* 84:292-299.